2. Care should be exercised in the selection of patients to prevent the possibility of causing the person who has not been dependent on heroin to become dependent on methadone.
3. There should be continued evaluation of the long-term effectiveness of methadone programs for persons who are stabilized on an inpatient or an ambulatory outpatient basis.
4. Where feasible, staff members of new methadone maintenance programs should be trained in this technique in an established effective program.
5. Continuing research is essential particularly with reference to:
   a. the use of properly trained practicing physicians as an extension of organized methadone maintenance programs in the management of those patients whose needs for allied services are minimal. These patients should remain in contact with the methadone maintenance program for periodic evaluation, including urine testing.
   b. the role of methadone maintenance in the treatment of heroin dependent patients under age 18 years.
   c. the use of methadone maintenance in combination with other approaches to the treatment of morphine type dependence.

Methadone maintenance is not feasible in the office practice of private physicians. The individual physician cannot provide all of the services for the various therapeutic needs of the patient. The individual physician also is not in a position to assure control against redistribution of the drug into illicit channels, to maintain control of doses, or to establish the elements for proper evaluation of the treatment. Practicing physicians, however, should cooperate with methadone maintenance programs in their communities and offer whatever services they may be capable of providing.

Highlights of Auscultation In Congenital Heart Disease

Part II

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Material Supplied by the American Heart Association

The Pulmonary Orifice. The midsystolic murmur of isolated congenital valvular pulmonic stenosis is typically loudest in the second left intercostal space and radiates upward and to the left. The length of the murmur varies directly with the degree of obstruction so that in mild pulmonic stenosis the murmur ends before both components of the second heart sound, whereas in severe pulmonic stenosis the murmur goes through aortic closure but necessarily ends before the delayed soft or inaudible sound of pulmonary valve closure. The murmur is introduced by a pulmonic ejection sound which often distinctively waxes with expiration and wanes with inspiration. Severe obstruction is associated with powerful right atrial contraction which distends the right ventricle in presystole and in so doing generates an atrial or fourth heart sound. Specific attention should be called to the systolic murmur that accompanies stenosis of the pulmonary artery and its branches, a congenital malformation that often follows maternal rubella. These murmurs are widely distributed in the right chest, axilla, and back, and must be sought by auscultation at nonprecordial sites during quiet respiration.

When one speaks of pulmonary regurgitation, the high frequency blowing Graham Steell mur-

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Part II of a two-part article. Part I appeared in the May issue.

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mur comes to mind. However, pulmonary regurgitation may occur without pulmonary hypertension when there is a congenital or acquired anatomic defect of the valve itself, and this murmur differs from that of Graham Steell. The murmur begins at an interval after the second heart sound, is crescendo-decresendo in shape, ends well before the next first heart sound, and is low to medium pitched since a low diastolic pressure in the pulmonary trunk results in a low rate of regurgitant flow.

The Atrial Septum. Atrial septal defect can be one of the most readily diagnosed congenital anomalies of the heart although from an auscultatory point of view, the malformation is often overlooked because of the relatively inconspicuous murmur. It should be borne in mind that the *defect itself* is acoustically silent and the shunt is diastolic; when the right ventricle ejects the large stroke volume accumulated in diastole, a relatively short grade 2-3 pulmonic systolic murmur is generated. The right ventricle takes longer to expel its large stroke volume so the second component of the second heart sound (pulmonary closure) is delayed; furthermore, the physiology of the circulation in atrial septal defect results in *fixed* splitting of the second heart sound which means that the split remains unchanged during respiration.

The soft murmur of atrial septal defect may be mistaken for an innocent systolic murmur, especially in children. However, the usual innocent murmur is a vibratory, buzzing, pure to medium frequency event that is best heard along the lower sternal edge and toward the apex and is accompanied by *normal* splitting of the second heart sound. In addition, *complete right bundle branch block* is associated with *wide* splitting of the second heart sound but usually not with *fixed* splitting and hence can be distinguished from the wide fixed splitting of the atrial septal defect.

The Ventricular Septum. The typical holosystolic left sternal edge murmur of ventricular septal defect is well known. Progressive pulmonary hypertension decreases the left to right shunt and shortens the murmur; when the shunt is reversed (Eisenmenger's Complex), the murmur through the defect is abolished. It is important to recognize that an early systolic murmur can *also* occur with *very small* nonpulmonary hypertensive ventricular septal defect in which the shunt is interrupted in latter systole. Such murmurs are soft, pure, high frequency, and quite localized at the mid to lower left sternal edge; as time goes on these murmurs may disappear because of spontaneous closure of the defect.

Fallot's tetralogy—the commonest cyanotic congenital cardiac above age 4 years—has been taken to represent a large ventricular septal defect upon which varying degrees of pulmonic stenosis are imposed. Progressive right ventricular outflow obstruction decreases the left to right interventricular shunt and shortens and finally abolishes the holosystolic murmur leaving an isolated midsystolic murmur of pulmonic stenosis. As obstruction increases further, right ventricular blood is shunted through the ventricular septal defect into the aorta; accordingly, pulmonary flow decreases, cyanosis increases, and the pulmonic stenotic murmur progressively shortens and softens disappearing completely with pulmonary atresia.

The Great Vessels. When an uncomplicated patent ductus arteriosus joins the great vessels, a characteristic continuous machinery murmur peaks around the second heart sound and is maximal at the left base. Several words of caution are appropriate. Occasionally a loud venous hum in young children transmits below the clavicles and is mistaken for a patent ductus arteriosus; this error can be avoided by compressing the deep jugular veins, a maneuver that abolishes the hum. In large patent ductus, progressive pulmonary hypertension decreases the left to right shunt so that the continuous murmur shortens, then becomes systolic, and when the shunt is reversed, disappears entirely. At this point, the recognition of patent ductus does not depend upon auscultation; instead, the presence of differential cyanosis (blue toes and pink fingers) makes the diagnosis.

The Myocardium

Hypertrophy. Increased force of atrial contraction usually distends a hypertrophied ventricle in presystole. Atrial or fourth heart sounds accompany the presystolic distention and are useful signs of hypertrophy such as in aortic stenosis or systemic hypertension on the left side and pulmonic stenosis or pulmonary hypertension on the right. In the pulmonary hypertension of emphysema, the atrial sound is often heard in the epigastrium since all of the heart sounds—including a loud pulmonary closure sound—are damped because of the large anteroposterior chest dimensions.

Failure. Third heart sounds are physiologic in children and young adults but pathologic in older subjects. Ventricular failure is a common cause of abnormal third heart sounds and should be specifically sought with light touch of the stethoscopic bell in all patients in whom heart failure is suspected. It is a point of interest that the effect of cardiac infarction on the left ventricular myocardium commonly results in the need for an increased distending force that is provided by augmented atrial contraction; atrial or fourth heart sounds are prevalent in this context.

An ischemic left ventricle may take longer than normal to eject so aortic valve closure may fall *after* pulmonary closure causing reversed or paradoxical splitting of the second heart sound. The commoner cause of paradoxical splitting however is left bundle branch block or a right ventricular pacemaker which is its electrical equivalent.

Constriction. Myocardial constriction—as in constrictive pericarditis—results in high atrial pressures and rapid flow into nondistensible ventricles. Under these circumstances, loud early third heart sounds occur and have been called "early diastolic sounds" of constrictive pericarditis. Similar sounds occur in the restrictive form of primary myocardial disease.

Summary. Modern instrumentation has not eclipsed the need for sophisticated auscultation. On the contrary, clinical research has gone far in clarifying the meaning of auscultatory events in acquired and congenital heart disease and has increased the value of the stethoscope as a clinical tool.

HOME TESTS FOR DEAFNESS MAY BE TOO LOUD

"Typically the parent of a deaf child senses that something is wrong in the first year of life. Then when speech would normally begin but doesn't, suspicions increase. . . . Parents will quite often bang pots together behind the child or take a hammer and hit a frying pan. Frequently, the resultant sounds are so loud or the vibrations are so great that even the child with a profound hearing loss jumps. The child may be responding to the vibrations or to the sound; but remember that a child is educationally and socially deaf when he is unable to hear speech sounds well enough to understand them. It is this kind of a hearing loss which must be diagnosed. The child may be able to hear airplanes, trucks, and banging pots and pans; but if he can't understand speech sounds, for practical purposes he's deaf. By these criteria of deafness, home tests obviously miss a great deal of deafness."

—McCay Vernon, PH.D., Chicago

Extracted from *Audio-Digest Otorhinolaryngology,* Vol. 2, No. 14, in the Audio-Digest Foundation's subscription series of tape-recorded programs. For subscription information: 619 S. Westlake Ave., Los Angeles, Ca. 90057.